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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/603,141	06/24/2003	Keith D. Allen	R-976	2357

26619 7590 10/14/2004

DELTAGEN, INC.
1031 Bing Street
San Carlos, CA 94070

EXAMINER	
HAMA, JOANNE	

ART UNIT	PAPER NUMBER
1632	

DATE MAILED: 10/14/2004

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	10/603,141	ALLEN, KEITH D.	
	Examiner	Art Unit	
	Joanne Hama, Ph.D.	1632	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *24 June 2003*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-9* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-9* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *24 June 2003* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date *none*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

This Application was filed June 24, 2003 and claims priority to U.S. Provisional Application No. 60/391,185, filed June 24, 2002.

Claims 1-9 are pending.

Claim Objection

Claim 1 should spell out CXCR6, when first presented.

Claim Rejections - 35 USC § 101and 112

35 U.S.C. 101 reads as follows:

> Whoever invents or discovers any new and useful process, machine, manufacture, or composition of matter, or any new and useful improvement thereof, may obtain a patent therefor, subject to the conditions and requirements of this title.

Claims 1-9 are rejected under 35 U.S.C. 101 because the claimed invention is not supported by either a specific and substantial asserted utility or a well-established utility.

According to MPEP 2107.01, concerning "specific utility":

> "A 'specific utility' is *specific* to the subject matter claimed. This contrasts with a *general* utility that would be applicable to the broad class of the invention. Office personnel should distinguish between situations where an applicant has disclosed a specific use for or application of the invention and situations where the applicant merely indicates that the invention may prove useful without identifying with specificity why it is considered useful.... A general statement of diagnostic utility, such as diagnosing an unspecified disease, would ordinarily be insufficient absent a disclosure of what condition can be diagnosed."

The specification states that, "given the importance of GPCRs, particularly chemokine receptors such as CXCR6, a clear need exists for the elucidation of their

functions, which information can be used in preventing, ameliorating or correcting dysfunctions or diseases associated therewith (page 3, lines 19-22)." However, the mice comprising a homozygous disruption in CXCR6 are not described in the specification as having any condition or disease associated with the lack of CXCR6 expression. The Example section of the specification describes a series of experiments which have been used to identify any phenotypes that the CXCR6 disrupted mice may have. According to the specification, the CXCR6 disrupted mice were examined for behavioral and physiological differences from wild type mice. CXCR6 disrupted mice were examined physiologically via a Physical Examination (Example 3), Necropsy (Example 4), Histopathological Analysis (Example 5), Hematological Analysis (Example 7), Serum Chemistry (Example 8), Densitometric Analysis (Example 9), Embryonic Development (Example 10), Fertility (Example 11), Cytoflurometric Analyses (Example 12), Metabolic Screens (Example 13), Pain and Noiciception (Example 14), Cutaneous Allergy (Example 15). The CXCR6 disrupted mice were examined for behavioral defects via the Rotarod Test, the Startle Test, the Hot Plate Test, the Tail Flick Test, the Open Field Test, the Metrazol Test, the Tail Suspension Test (Example 6). However, none of the tests listed described any differences between the CXCR6 disupted mouse and a wild type mouse. There is no evidence on record that the claimed mice, cells, construct, and method of making have any utility that the skilled artisan would regard as specific, "where (the) applicant has disclosed a specific use."

According to MPEP 2107.01, concerning "substantial utility":

> "A 'substantial utility' defines a 'real world' use. Utilities that require or constitute carrying out further research to identify or reasonably confirm a 'real world'

> context of use are not substantial utilities.... the following are examples of situations that require or constitute carrying out further research to identify or reasonably confirm a 'real world' context of use and therefore, do not define 'substantial utilities':
>
> A) Basic research such as studying the properties of the claimed product itself or the mechanisms in which the material is involved;
> B) A method of treating an *unspecified* disease or condition:
> C) A method of assaying for or identifying a material that itself has no specific and/or substantial utility;
> D) A method of making a material that itself has no specific, substantial, and credible utility; and
> E) A claim to an intermediate product for use in making a final product that has no specific substantial and credible utility.

While as stated in the specification, "given the importance of GPCRs, particularly chemokine receptors such as CXCR6, a clear need exists for the elucidation of their functions, which information can be used in preventing, ameliorating or correcting dysfunctions or disease associated therewith," the specification does not disclose that the homozygous CXCR6 disrupted mice have any phenotype. The specification does not teach one skilled in the art how to use the homozygous CXCR6 disrupted mice to study a disease or how to use the mice as test subjects in a screen for therapeutics. These mice are not a readily available utility because all homozygous and heterozygous CXCR6 disrupted mice have no phenotype. The homozygous CXCR6 disrupted mice require "further research to identify or reasonably confirm a 'real world' context of use," and demonstrate three MPEP examples that do not define "substantial utilities" of these mice: "A) Basic research such as studying the properties of the claimed product itself or the mechanisms in which the material is involved," "B) A method of treating an *unspecified* disease or condition," and "D) A method of making a material that itself has

no specific, substantial, and credible utility." More specifically, the specification does not state that the CXCR6 disrupted mice have any phenotype. Because the specification does not demonstrate "substantial utility" for the claimed mice, the construct, cells, and the method of making do not have a substantial utility.

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 1-9 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter, which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

The Background of the Invention (pages 1-3) teaches the isolation and the identification of CXCR6. CXCR6 was isolated by Liao et al. by RT-PCR on tumor-infiltrating lymphocyte lines with degenerate primers based on sequences of known chemokine receptors. Liao et al. named the isolated gene STRL33. Using an expression cloning strategy with a T-cell cDNA library, Deng et al. isolated an identical cDNA, which was named BONZO. The STRL33/BONZO sequence shared a 25-30% amino acid identity with other chemokine receptors. Northern blot analysis detected a 2.1-kb transcript in spleen, thymus, small intestine, and to a lesser extent in peripheral blood leukocytes, prostate, and colon. A 2.6-kb transcript was detected in placenta. Studies have shown that CXCR6 may be a coreceptor with CD4 for fusion with

macrophage-tropic HIV-1. Another study has shown that CXCR6 may function in interactions between dendritic cells and T-cells and in regulating T-cell migration in the splenic red pulp. These studies suggested roles that CXCR6 may have in T-cells.

Example 2: Expression Analysis describes the expression pattern of mRNA and β-gal in wild type and heterozygous mice that have undergone homologous recombination for the CXCR6 locus. The results generated from the RT-PCR, done on wild type mice indicates which tissues and organs are positive for the transcript being detected. While it is not clearly stated, it is assumed that the mRNA being detected in the RT-PCR assay is CXCR6. During the generation of CXCR6 disrupted mice, one way of ensuring that the correct gene had been targeted during homologous recombination would be to check for expression of a reporter gene. If homologous recombination occurred, then the reporter gene should express in a pattern similar to that of the endogenous gene that was targeted. According to the RT-PCR results, RNA transcripts were strongest in the spleen and lymph nodes. According to the β-gal results, β-gal was detected in the urinary bladder and skin. In the urinary bladder, a few cells in the lamina propria showed moderate staining. A small number of epidermal cells also showed X-gal staining. One issue one skilled in the art would wonder would be if CXCR6 was in fact targeted since the RT-PCR results and the β-gal results are different. Based on the localization differences between the RT-PCR and the β-gal results, the possibility exists that another CXCR family member could have been targeted. For this reason, the specification has not adequately taught how to make CXCR6 disrupted mice. While the β-gal results indicate that few cells in the skin and

urinary bladder had very intense staining, the specification did not teach what these cells were. An indication that these intensely staining cells were T-cells might give one skilled in the art more confidence that it is likely that a CXCR family member was targeted. However, the specification does not provide guidance that the targeted gene was CXCR6 and not another family member.

The CXCR6 disrupted mice are also not enabled because the specification does not describe how to use homozygous disrupted mice that have no phenotype. According to the results provided in the specification, the specification provides no guidance on how to use CXCR6 mice as a model for a disease related to CXCR6 disruption, or how to use the mice as a test subject to screen for therapeutics treating a disease related to CXCR6.

The claims are broad and encompass other kinds of mice. No guidance has been given in the specification as to how to use these mice. The claimed mice also encompass chimeric mice where the disruption is only in some of the cells of the mouse (claim 7). These mice are not enabled because the specification has not provided guidance necessary to reproducibly make such mice where the mice have an enabled use. The method of making genetic mosaic mice is such that each resulting chimera is comprised of a different, unpredictable ratio of cells of various genotypes. This ratio cannot be predetermined. Therefore, the phenotype of chimeric animals is not only dependent upon the genotype of the cells (which is unpredictable as set forth by the state of the art) but is also dependent upon the spatial distribution of the cells and their relative population size.

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 1-9 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The final Written Description Examination guidelines that were published on January 5, 2001 (66 FR 1099; available at http://www.uspto.gov/web/menu/current.html#register).

Vas-Cath Inc. v. Mahurkar, 19USPQ2d 1111 (Fed. Cir. 1991), clearly states that "applicant must convey with reasonable clarity to those skilled in the art that, as of the filing date sought, he or she was in possession *of the invention*. The invention is, for purposes of the 'written description' inquiry, *whatever is now claimed.*" Vas-Cath Inc. v. Mahurkar, 19USPQ2d at 1117. The specification does not "clearly allow persons of ordinary skill in the art to recognize that [he or she] invented what is claimed." Vas-Cath Inc. v. Mahurkar, 19USPQ2d at 1116.

While the specification describes that a murine CXCR6 cDNA corresponds to SEQ ID NO: 1, the specification fails to adequately describe the characteristics that

uniquely define the mouse CXCR6 sequence. The claimed invention as a whole is not adequately described if the claims require essential or critical elements which are not adequately described in the specification and which are not conventional in the art as of Applicants effective filing date. Possession may be shown by actual reduction to practice, clear depiction of the invention in a detailed drawing, or by describing the invention with sufficient relevant identifying characteristics (as it relates to the claimed invention as a whole) such that a person skilled in the art would recognize that the inventor had possession of the claimed invention. Pfaff v. Wells Electronics, Inc., 48 USPQ2d 1641, 1646 (1998). In the instant case, the Applicant has stated that mouse CXCR6 cDNA is SEQ ID NO. 1 and that the corresponding amino acid sequence is SEQ ID NO. 2. However the specification fails to describe the relevant identifying characteristics of mouse CXCR6 cDNA. As a result, it is difficult for one skilled in the art to determine whether the CXCR6 disrupted mice described in the Application have a gene disruption in the CXCR6 gene. One major issue that the written description would resolve is how to discriminate one's gene of interest from other family members.

There are a few situations that result from a lack of written description of the mouse CXCR6 cDNA. First, the specification does not teach what CXCR6 cDNA encodes. On one level, this would entail describing protein domains that are common to all other CXCR family members. On another level, this would entail describing what protein regions of CXCR6 are unique that it does not function like other CXCR family members. This is important during the generation of gene targeting. One skilled in the art would not design a targeting construct made against a domain common to other

family members. It is conceivable that a targeting construct made against a common domain has high probability it could target other family members that have that domain. Second, the specification does not teach ways to determine (e.g. assays) the function of CXCR6 in a cell or tissue. In addition to these experiments teaching one skilled in the art what features are unique to CXCR6 that discriminate it from its family members, the experiments should, ideally, complement the phenotypes seen in the animal model. These assays can be used to define the mechanism involved in mediating the phenotype. Admittedly, there are times that the animal model presents an unexpected phenotype. However, if one skilled in the art has defined criteria that is unique for one's gene of interest, the issue is less of "was this animal made correctly?" and more, "what is the biological basis of this?" The skilled artisan cannot envision how to discriminate CXCR6 from all the possible variant CXCR family members, and therefore conception is not achieved until reduction to practice has occurred, regardless of the complexity or simplicity of the method used. Adequate written description requires more than a mere statement that it is part of the invention and reference to a potential method of identifying it. See *Fiers v. Revel*, 25 USPQ2d 1601, 1606 (Fed. Cir. 1993) and *Amgen Inc. v. Chugai Pharmaceutical Co. Ltd.*, 18 USPQ2d 1016 (Fed. Cir. 1991).

One cannot describe what one has not conceived. See *Fiddes v. Baird*, 30 USPQ2d 1481, 1483. In *Fiddes*, claims directed to mammalian FGF's were found to be unpatentable due to lack of written description for that broad class. The specification provided only the bovine sequence.

Therefore, CXCR6 disruption in a mouse does not meet the written description provision of 35 U.S.C. §112, first paragraph. Applicant is reminded that *Vas-Cath* makes clear that the written description provision of 35 U.S.C. §112 is severable from its enablement provision (see page 1115).

Applicants attention is drawn to the decision of *The Regents of the University of California v. Eli Lilly and Company* (CAFC, July 1997) wherein it was stated:
In claims involving chemical materials, generic formulas usually indicate with specificity what the generic claims encompass. One skilled in the art can distinguish such a formula from others and can identify many of the species that the claims encompass. Accordingly, such a formula is normally an adequate written description of the claimed genus. In claims to genetic material, however, a generic statement such as "vertebrate insulin cDNA" or "mammalian cDNA," without more, is not an adequate written description of the genus because it does not distinguish the claimed genus from others, except by function. It does not specifically define any of the genes that fall within its definition. It does not define any structural features commonly possessed by members of the genus that distinguish them from others. One skilled in the art therefore cannot, as one can do with a fully described genus, visualize or recognize the identity of the members of the genus. A definition by function, as we have previously indicated, does not suffice to define the genus because it is only an indication of what the gene does, rather than what it is. See *Fiers,* 984 F.2d at 1169-71, 25 USPQ2d at 1605-06 (discussing *Amgen*). It is only a definition of a useful result rather than a definition of what it achieves as a result. Many such genes may achieve that result. The description

requirement of the patent statute requires a description of an invention, not an indication of a result that one might achieve if one made that invention. *See In re Wilder*, 736 F.2d 1516, 222 USPQ 369, 372-373 (Fed. Cir. 1984) (affirming rejection because the specification does "little more than outlin[e] goals appellants hope the claimed invention achieves and the problems the invention will hopefully ameliorate."). Accordingly, naming a type of material generally known to exist, in the absence of knowledge as to what that material consists of, is not a description of that material.

Because Applicants have failed to provide an adequate written description of the materials used in the compositions and methods claimed and because there is no evidence that Applicants possessed any CXCR6 beyond that disclosed and/or known in the prior art, the rejected claims fail to meet the written description requirement under 35 U.S.C. 112, first paragraph. Applicant is reminded that *Vas-Cath* makes clear that the written description provision of 35 U.S.C. §112 is severable from its enablement provision (see page 1115).

Claim Rejections - 35 USC § 112

The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

Claims 1, 3, 6, 8, 9 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claim 6 is incomplete since guidance is not provided on producing a mouse whose genome comprises a CXCR6 disruption. In claim 6(b), a viable transgenic mouse does not develop from a murine stem cell implanted in a pseudopregnant mouse. Mouse ES cells by themselves cannot develop into a mouse; they must be inserted into a blastocyst and then implanted into the uterus of a pseudopregnant mouse. Claim 6 is also incomplete because the transgenic mouse that is born is mosaic. However, the qualifying statement about the mouse, "wherein the transgenic mouse lacks production of functional CXCR6 and exhibits a phenotypic abnormality" implies that the whole mouse has a gene disruption.

Claim 3 improperly depends on claim 1. Claim 1 states, "wherein the transgenic mouse lacks production of functional CXCR6." Claim 3 states, "wherein the disruption in the endogenous CXCR6 gene is heterozygous." The dependence is improper because the mice in claim 1 have no endogenous expression of CXCR6, where the mice in claim 3 has endogenous CXCR6 expression. The claims are unclear and confusing because it appears that these are two kinds of mice with different levels of endogenous CXCR6 expression.

Claim 1 and 6 are vague because "abnormality" is a relative term. The problem arises because in many situations, there is a range of manifested phenotypes and they are considered "normal" depending on what the parameters are. Depending on what is being measured, a few millimeters of difference in length is significant for one body part and not significant for another. Furthermore, it cannot always be assumed that if one person considers a difference between a knockout and a wild type animal that another

person will necessarily agree that there is a difference. Claims 2, 3, 4, and 7 depend from claims 1 and 6.

"Homologous" in claim 8 is vague. Claim 9 depends from claim 8. It is not clear what is meant by "homologous" because there are different interpretations of "homologous." For example, if sequence A is 200 bases long and sequence B is 200 bases long and both have the same sequence, then sequence A and B are 100 percent homologous to each other. However, if sequence A is 500 bases long and sequence B is 200 bases long and sequence B matches a region in sequence A perfectly, sequence B would be considered to be 100 percent homologous to A, but A is not 100 percent homologous to B. The use of the word "homologous" also brings up issues addressed in the Written Description section above. While the specification teaches that homologous "denotes a characteristic of a DNA sequence having at least about 70 percent sequence identity... (page 8, lines 10-11)," the question then arises, how does one discriminate between CXCR6 and its family members that have about 70 percent homology to CXCR6? How does one know for certain that if a construct made for homologous recombination, which has 70 percent homology to the target site, will actually hit the intended target and not a family member?

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless –

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

Claims 1-9 are rejected under 35 U.S.C. 102(b) as being anticipated by Unutmaz et al. (2000, The Journal of Immunology, 165: 3284-3292).

Unutmaz et al. teach that little was known about the cell subset-specific expression and regulation of BONZO (another name for CXCR6). One way of identifying an *in vivo* function is to develop a knockout model. Unutmaz et al. teach how a homozygous BONZO (another name for CXCR6) disrupted mouse was made. A knockin targeting construct was made, where the GFP reporter gene and the neomycin selection marker were inserted into a site between the 5' untranslated region (UTR) and a genomic region downstream of the open reading frame (ORF) (page 3286, first column, "gene targeting in embryonic stem cells and generation of mice"). The resulting mouse was a knockout of BONZO, but a knockin of GFP in the BONZO locus. Since the GFP was under the control of the BONZO promoter, GFP would be expressed in the same cells that would normally express BONZO. The homozygous BONZO disrupted mouse was analyzed for phenotypes. The homozygous BONZO disrupted mouse developed no detectable phenotypes. However, the authors suggest that this may be due to redundancy in the chemokine receptor system. The authors stress that their studies have not be exhaustive and will be carrying out more detailed analyses on the mice. The authors also stress that it may be possible the phenotypes in the BONZO disrupted mice only are obvious in studies where they are challenged with pathogens or in disease models (page 3291, first column, third paragraph).

Unutmaz et al. anticipate the homozygous CXCR6 disrupted mouse made by the Applicant. For reasons of inherency, Unutmaz et al. also anticipate the phenotypes associated with CXCR6 disruption in the mouse.

Conclusion

No claims allowed.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Joanne Hama, Ph.D. whose telephone number is (571) 272-2911. The examiner can normally be reached on Monday-Friday 9:00-5:00.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Amy Nelson, Ph.D. can be reached on (571) 272-0804. The fax phone number for the organization where this application or proceeding is assigned is 703-872-9306.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

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